8320157D.TRN **KEEP WITH FILE 08320157D.TRN**
November 13, 1996

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 ********************************
FILE 'USPAT' ENTERED AT 07:10:44 ON 13 NOV 96

 ********************************
 *       WELCOME TO THE         *
 *    U.S. PATENT TEXT FILE     *
 ********************************

=> s (cdn? or cdi?) not (cdna?)
      4929 CDN?
      1259 CDI?
      4679 CDNA?
L1     1479 (CDN? OR CDI?) NOT (CDNA?)

=> s apoptosis
L2       71 APOPTOSIS

=> s l1 and l2
L3        1 L1 AND L2

=> d l3 cit ab

1. 5,559,038, Sep. 24, 1996, Gas chromatography/mass spectrometry
determination of oxidized sulfhydryl amino acids; J. Fred Kolhouse, et
al., 436/86, 120, 161, 173, 174, 178 [IMAGE AVAILABLE]

US PAT NO:    5,559,038 [IMAGE AVAILABLE]          L3: 1 of 1

ABSTRACT:
Method for quantifying the amounts of oxidized sulfhydryl amino acids,
particularly cysteine sulfinic acid, cysteic acid, homocysteine sulfinic
acid and homocysteic acid, for example, in biological samples are
provided. The oxidized sulfhydryl amino acids are measured by gas
chromatography/mass spectrometry after derivatization, preferably using
stable isotope internal standards. Deviation from normal individuals'
levels of oxidized sulfhydryl amino acids in serum are indicative of
certain neuropsychiatric disorders, the biochemical origin of which can
be diagnosed using methods disclosed herein, particularly when relative
levels of these and/or other sulfur amino acids are measured.

=> quit
ALL L# QUERIES AND ANSWER SETS ARE DELETED AT LOGOFF
LOGOFF? (Y)/N/HOLD:y

U.S. Patent & Trademark Office LOGOFF AT 07:11:40 ON 13 NOV 96
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8320157B.TRN **KEEP WITH FILE 08320157B.TRN**
November 13, 1996

FILE 'HOME' ENTERED AT 07:02:40 ON 13 NOV 96

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COST IN U.S. DOLLARS SINCE FILE TOTAL
ENTRY SESSION
FULL ESTIMATED COST 0.15 0.15

FILE 'MEDLINE' ENTERED AT 07:03:21 ON 13 NOV 96

FILE 'BIOSIS' ENTERED AT 07:03:21 ON 13 NOV 96
COPYRIGHT (C) 1996 BIOSIS(R)

FILE 'EMBASE' ENTERED AT 07:03:21 ON 13 NOV 96
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FILE 'CAPLUS' ENTERED AT 07:03:21 ON 13 NOV 96
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(FILE 'HOME' ENTERED AT 07:02:40 ON 13 NOV 96)

FILE 'MEDLINE, BIOSIS, EMBASE, CAPLUS' ENTERED AT 07:03:21 ON 13 NOV 96
L1 4448 FILE MEDLINE
L2 4598 FILE BIOSIS
L3 4145 FILE EMBASE
L4 7135 FILE CAPLUS
TOTAL FOR ALL FILES
L5 20326 S (CDN? OR CDI?) NOT (CDNA)
L6 7541 FILE MEDLINE
L7 12113 FILE BIOSIS
L8 8406 FILE EMBASE
L9 7502 FILE CAPLUS
TOTAL FOR ALL FILES
L10 35562 S APOPTOSIS
L11 15 FILE MEDLINE
L12 18 FILE BIOSIS
L13 17 FILE EMBASE
L14 18 FILE CAPLUS
TOTAL FOR ALL FILES
L15 68 S L5(P)L10
L16 446367 FILE MEDLINE
L17 373775 FILE BIOSIS
L18 327137 FILE EMBASE
L19 216157 FILE CAPLUS
TOTAL FOR ALL FILES
L20 1363436 S ANTIBOD?
L21 29 DUP REM L15 (39 DUPLICATES REMOVED)

=> s l15 not (cdnas)
L22 1 FILE MEDLINE
L23 2 FILE BIOSIS
L24 1 FILE EMBASE

L25 1 FILE CAPLUS

TOTAL FOR ALL FILES
L26 5 L15 NOT (CDNAS)

=> dup rem l26
PROCESSING COMPLETED FOR L26
L27 3 DUP REM L26 (2 DUPLICATES REMOVED)

=> d l27 1- bib ab

L27 ANSWER 1 OF 3 BIOSIS COPYRIGHT 1996 BIOSIS
AN 96:191087 BIOSIS
DN 98747216
TI Differentiation of U-937 promonocytic cells with mitomycin C or cis-diamminedichloroplatinum II.
AU Ballester A; Perez C; Aller P; Mata F
CS Dep. Bioquimica Biologia Molecular I, Fac. Biol., Univ. Complutense, 28040 Madrid, Spain
SO International Journal of Cancer 65 (6). 1996. 791-795. ISSN: 0020-7136
LA English
AB Administration of 0.3 mu-M mitomycin C (MMC) or 2.0 mu-M cis-diamminedichloroplatinum II (CDDP) decreased the growth activity and induced the differentiation of U-937 human promonocytic cells, as shown by nitroblue tetrazolium reduction and an increase in surface expression of the leukocyte integrins ***CDIIb*** /CD18 and ***CDIIc*** /CD18. Expression of these differentiation markers started to be significant at 48 hr of treatment. These concentrations resulted in little cell damage (determined by Trypan blue exclusion) and slightly induced ***apoptosis*** (determined by DNA degradation and changes in nuclear morphology). The treatments induced a transient increase in c-fos and c-jun mRNA levels, with maximum values at 1-6 hr; a transient increase in collagenase mRNA level, with a maximum value at 48 hr; and a progressive increase in vimentin and lamin A and C mRNA levels. These changes were qualitatively similar to those produced by 12-O-tetradecanoylphorbol 13-acetate. CDDP and MMC also caused a transient increase of total AP-I binding activity, as determined by gel retardation assays. The drugs produced an early transient activation (3-6 hr) of membrane-bound protein kinase C, followed by a later activation (48 hr) of both the membrane and the cytosolic enzyme. These results suggest that protein kinase C and AP-I-dependent gene expression could be involved in myeloid cell differentiation by alkylating agents.

L27 ANSWER 2 OF 3 BIOSIS COPYRIGHT 1996 BIOSIS DUPLICATE 1
AN 96:311792 BIOSIS
DN 99034148
TI The dual role of S-nitrosoglutathione (GSNO) during thymocyte ***apoptosis*** .
AU Sandau K; Bruene B
CS Univ. Erlangen-Nuernberg, Fac. Med., Dep. Med. IV-Experimental Div., 91054 Erlangen, Germany
SO Cellular Signalling 8 (3). 1996. 173-177. ISSN: 0898-6568
LA English
AB Nitric oxide (NO) is known to regulate redox-sensitive signalling

pathways in physiology and pathophysiology. Depending on its concentration, the NO-releasing compound S-nitrosoglutathione (GSNO) causes negative and positive regulation of thymocyte ***apoptosis*** . At levels below 0.6 mM, GSNO produces deoxyribonucleic acid (DNA) laddering, which is inhibited by activation of protein kinase C(PKC), cycloheximide treatment, and calcium chelation. Higher concentrations of the NO donor (1-2 mM) suppress thymocyte ***apoptosis*** initiated by the classical agonist dexamethasone. Inhibition of ***apoptosis*** by NO is analogous to the action of the thiol-blocking compound N-ethylmaleimide (NEM) and the glutathione-S-transferase substrate 1-chloro-2,4-dinitrobenzene (***CDNB***). Inhibition of ***apoptosis*** results from thiol modification of critical proteins in response to NO treatment. Depending on the concentration, GSNO can be involved either in toxic or in protective signalling in thymocyte biology.

L27 ANSWER 3 OF 3 MEDLINE
AN 95370107 MEDLINE
TI Bcl-2: prevention of ***apoptosis*** as a mechanism of drug resistance.
AU Reed J C
CS La Jolla Cancer Research Foundation, Cancer Research Center, California, USA..
SO HEMATOLOGY/ONCOLOGY CLINICS OF NORTH AMERICA, (1995 Apr) 9 (2) 451-73. Ref: 89
Journal code: HEO. ISSN: 0889-8588.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 9511
AB Programmed cell death (also known as ***apoptosis***) plays an essential role in tissue homeostasis, where it ensures that new cell production in the body is offset by a commensurate rate of cell loss. Defects in the genetic pathway that regulate the cell death process can figure prominently in the origins of cancer and also in problems with cancer treatment. Eventually, it may be possible to develop novel treatments for cancer that specifically seek to modulate the physiologic cell death pathway as opposed to nearly all currently available drugs, which are intended to interfere with some aspect of the cell division cycle.

=> file wpids

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		35.41	35.56

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SINCE FILE SESSION	TOTAL
CA SUBSCRIBER PRICE		-1.85	-1.85

FILE 'WPIDS' ENTERED AT 07:08:56 ON 13 NOV 96

FILE LAST UPDATED: 11 NOV 96 <961111/UP>
>>>UPDATE WEEKS:
MOST RECENT DERWENT WEEK 9645 <199645/DW>
DERWENT WEEK FOR CHEMICAL CODING: 9634
DERWENT WEEK FOR POLYMER INDEXING: 9642
DERWENT WORLD PATENTS INDEX SUBSCRIBER FILE, COVERS 1963 TO DATE
>>> DERWENT POLYMER INDEXING THESAURUS AVAILABLE IN FIELD /PLE <<<
>>> PATENT IMAGES AVAILABLE FOR PRINT AND DISPLAY <<<
>>> PATENTS CITATION INDEX AVAILABLE AS FILE DPCI <<<

=> s (cdn? or cdi?) not (cdna?)
2241 CDN?
190 CDI?
2206 CDNA?
L28 225 (CDN? OR CDI?) NOT (CDNA?)

=> s apoptosis
L29 141 APOPTOSIS

=> s l28 and l29
L30 1 L28 AND L29

=> d l30 1- bib ab

L30 ANSWER 1 OF 1 WPIDS COPYRIGHT 1996 DERWENT INFORMATION LTD
AN 95-215106 [28] WPIDS
DNC C95-099466
TI New nucleic acid sequences encoding ***Cdn*** ***apoptosis*** modulators - and related vectors, transformed cells, proteins and antibodies, useful for diagnosis and treatment e.g. of HIV infection, reperfusion injury etc..
DC B04 D16
IN BARR, P J; KIEFER, M C
PA (LXRB-N) LXR BIOTECHNOLOGY INC
CYC 20
PI WO 9515084 A1 950608 (9528)* EN 66 pp
AU 9513351 A 950619 (9540)
NO 9602188 A 960723 (9639)
EP 731636 A1 960918 (9642) EN
R: AT BE CH DE DK ES FR GB GR IE IT LI LU MC NL PT SE
FI 9602247 A 960722 (9642)
ADT WO 9515084 A1 WO 94-US13930 941130; AU 9513351 A AU 95-13351 941130; NO 9602188 A WO 94-US13930 941130, NO 96-2188 960529; EP 731636 A1 WO 94-US13930 941130, EP 95-904808 941130; FI 9602247 A WO 94-US13930 941130, FI 96-2247 960529
FDT AU 9513351 A Based on WO 9515084; EP 731636 A1 Based on WO 9515084
PRAI US 94-320157 941007; US 93-160067 931130
AB WO 9515084 A UPAB: 950721
Purified nucleic acid (I) encoding a ***Cdn*** is new. Also new is pure natural or recombinant ***Cdn*** protein.
USE - ***Cdn*** proteins are modulators of ***apoptosis*** which can be controlled by altering endogenous ***Cdn*** levels, either by increasing expression of a recombinant or endogenous gene or by admin. of ***Cdn*** protein.
Cdn can be used in all cases where superoxide dismutase is a suitable treatment e.g. HIV infection, autoimmune disease, cardiac or neuronal diseases, hepatitis, osteoporosis, shock or

proliferative disease (e.g. B cell lymphoma or eczema). Some partic. applications are ex vivo transfection of T cells with (I) before return to an HIV-positive subject to increase T cell survival time, in vivo transfection to prevent atherosclerosis, prevention of reperfusion injury in cerebral or myocardial infarct, as a pretreatment before admin. of cardiotoxic drugs, etc. Ab can be used to detect ***Cdn*** expression for diagnosis or monitoring; abnormal levels of ***Cdn*** are likely to indicate inappropriate ***apoptosis*** .

ADVANTAGE - Control of ***Cdn*** expression allows very specific modulation of ***apoptosis*** .

Dwg.0/11

=> d his

(FILE 'HOME' ENTERED AT 07:02:40 ON 13 NOV 96)

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L13 17 FILE EMBASE
L14 18 FILE CAPLUS
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L18 327137 FILE EMBASE
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L21 29 DUP REM L15 (39 DUPLICATES REMOVED)
L22 1 FILE MEDLINE
L23 2 FILE BIOSIS
L24 1 FILE EMBASE
L25 1 FILE CAPLUS
TOTAL FOR ALL FILES
L26 5 S L15 NOT (CDNAS)
L27 3 DUP REM L26 (2 DUPLICATES REMOVED)

FILE 'WPIDS' ENTERED AT 07:08:56 ON 13 NOV 96
L28 225 S (CDN? OR CDI?) NOT (CDNA?)
L29 141 S APOPTOSIS
L30 1 S L28 AND L29

=> quit h

COST IN U.S. DOLLARS	ENTRY	SINCE FILE SESSION	TOTAL
FULL ESTIMATED COST		3.53	39.09

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	ENTRY	SINCE FILE SESSION	SINCE FILE	TOTAL
CA SUBSCRIBER PRICE		0.00	-1.85	

SESSION WILL BE HELD FOR 60 MINUTES